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ENDURANCE OF EXTREMAL ACCELERATION INCREASED AS A RESULT OF EXPOSURE TO IONIZING RADIATION

B. I. Davydov

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ENDURANCE OF EXTREMAL ACCELERATION INCREASED AS A RESULT OF EXPOSURE TO IONIZING RADIATION[1]

B. I. Davydov

ABSTRACT

The author used 1690 white mice to investigate post-radiation resistance (doses ranged from 100-4000 R) to 3 min back-chest accelerations of 42-44 G. He found that 1-8 days after irradiation, experimental animals were more resistant to accelerations than control animals and that the degree of post-radiation resistance depended on radiation dose. He speculated that shifts in blood clotting function and cell membrane permeability during certain stages of radiation sickness might improve hemodynamic conditions for exposure to critical accelerations.

During more prolonged future space flights, the role of the radiation factor will undoubtedly grow. Therefore, a study of radiobiological problems during space flights is rendered important (refs. 1-3). /691*

Both in the theoretical and practical sense, the role of cosmic radiation in changes in cosmonaut reactivity to other flight factors commands interest. It is known that radiation substantially alters the functional condition of the organism (refs. 4,5). An understanding of the peculiarities of reactions of the irradiated organism to the effects of acceleration, vibration, weightlessness, etc., will permit the planning of normalizing measures.

The present report makes an attempt to assess the reaction of irradiated animals to acceleration. Here, attention was focused only on the quantitative aspect of the effect. To a lesser extent, the qualitative features of the reaction of the irradiated organism were considered.

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*Numbers given in margin indicate pagination in original foreign text.

[1]Submitted by Academician N. M. Sisakyan, 22 Jul 1965.

Experiments were conducted on 1690 mongrel white male mice. The degree of the reactivity of the animals was judged as a function of viability (or mortality) after exposure to critical magnitudes of accelerations. Such a method of investigation gives a general picture of the reactive condition of the animal during a period of extremal stimulation and also permits a qualitative evaluation of this condition. The use of an extremal stimulus can reveal those concealed and unstable compensatory mechanisms which would otherwise pass unnoticed during weak stimuli.

An RUM-11 with the following parameters was used for irradiation: Al 1 mm, Cu 0.5 mm, 180 kV, 10 mA. A "Khizotron" assembly was also used (Co^{60} gamma rays in doses of 100-4000 R; 13-18 R/min). From one to 45 days after irradiation, the animals were exposed to accelerations of 42-44 G for 3 min. with an acceleration buildup and deceleration duration of 50 sec. The centrifugal force was directed in a back-chest direction. Control animals were centrifuged simultaneously with irradiated animals.

Tests took place on a centrifuge with an arm radius of 0.31 m. It should be noted that considerable angular accelerations occurred under these conditions; accelerations were responsible for the mortality of 40-50 percent of the control animals. A deviation from this value was used as an indication of a positive or negative shift in the reactivity of irradiated animals.

Figure 1 gives the results of the experiment. Beginning with the first and second day after irradiation, the resistance of irradiated animals to critical accelerations was greater than the resistance of control animals. From the 8th day after irradiation, the resistance of animals to accelerations decreased. This lowered resistance of irradiated mice to critical accelerations coincided with the initial radiation mortality of the animals during this period. At doses of 200-4000 R, increased resistance to accelerations was noted for 1-6 days after irradiation. Later (9-45 days), the reaction of animals to acceleration was noted using lesser doses (< 700 R). At 200 R, lowered resistance to acceleration was noted and this was maintained up to the 45th day after irradiation.

The pathogenic mechanisms underlying this paradoxical phenomenon at first glance, e.g., increased resistance of irradiated animals to acceleration, cannot be fully assessed at the present time. Experimental data obtained from another study indicate that acceleration apparently is not a stress factor to the /692 fullest extent. For instance, it was shown that hypophysectomized rats less resistant to stressor effects were more resistant to positive accelerations (20 G) than control animals (ref. 6). It is interesting that during the latent period of radiation sickness, the resistance of animals to a mechanical stress increases (ref. 7). Also, the increased resistance of irradiated mice to accelerations indicates that acceleration is evidently not a stress factor in the classical sense. In our opinion, one of the possible reasons for increased resistance of irradiated animals to accelerations is apparently associated with shifts in the blood clotting system and the permeability of cell membranes occurring at certain stages of radiation sickness which could improve hemodynamic conditions during accelerations.

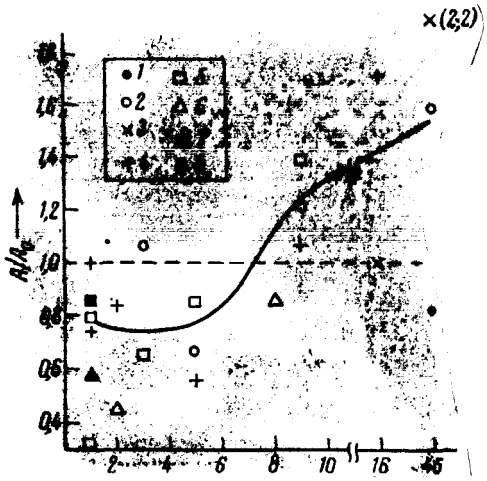

Figure 1. Reaction of mice to critical acceleration after exposure to ionizing radiation. A. Experimental mortality (percent); A_0- control mortality (percent). 1, 100; 2, 200, 250; 3, 300, 350; 4, 500; 5, 700; 6, 850; 7, 2000; 8, 4000 R.

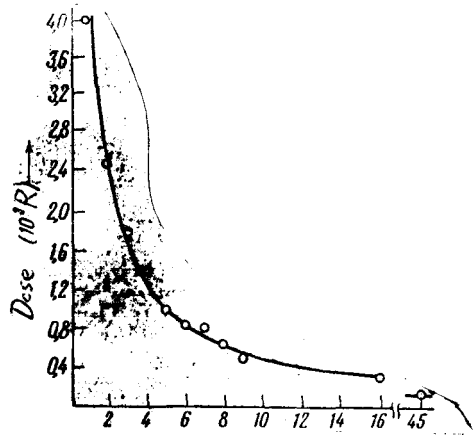

Figure 2. Dependence between the time after irradiation and the threshold dose where tolerance of critical acceleration by irradiated animals equals control tolerance.

The degree of the resistance in mice to accelerations is definitely dependent on the radiation dose. Using a method of mathematical extrapolation-interpolation, that dose of radiation where experimental tolerance equaled control tolerance was calculated. Figure 2 shows the results of this calculation. There is good coincidence between the computed data and an equation for a hyperbolic curve, $Dt=5\cdot10^3$, where D is the dose (in roentgens) and t is the time after irradiation (in days).

The asymptotic approximation of the horizontal portion of the curve suggests the presence of a threshold reaction in animals to this type of stress. In determining the minimum threshold dose of radiation as a function of acceleration, it is acceptable to use a time interval as the final point on the time scale in figure 2. This interval is equal to half the period of complete post-radiation recovery, during which 90 percent of the compensation of the reversible component of radiation sickness takes place. The following formula is given: $D_e = D_0[f-(1-f)e^{-\beta t}]$ (ref. 8), where D_e is the effective radiation dose, D_0 is the radiation dose, f is the irreversible component of radiation injury (in our case, 10 percent, f=0.1), t is the number of days, β is the rate /693 of recovery (for mice, 12.5 percent per day, i.e., β=0.125). The duration of half the period of complete post-radiation recovery is $\sim$ 19 days. The radiation dose yielded by the formula $Dt=5\cdot10^3$ for this period is 260 R, although, as shown in figure 2, at a later time (45 days after irradiation) the threshold dose obtained experimentally is $\sim$ 100 R. It is possible that this

disagreement is due to increased radiosensitivity of animals at the time of this experiment, or the irreversible component of radiation injury can be revealed as a function of tolerance of acceleration at doses of $\sim$ 100 R.. In our opinion, the latter hypothesis is unlikely, since at a dose of 100 R after 45 days, only the irreversible component of radiation equal to $\sim$ 10 R remains. Evidently, a more accurate threshold dose value (according to tolerance of extremal acceleration) for mice would be 250-260 R.

Earlier (ref. 4) it was found that the dependence between dose power and the development time of the initial reaction of the central nervous system is expressed by a hyperbolic function. It is known that the Weiss "Lapik" curve reflecting the excitability regularity of the most heterogeneous tissues during the action of an electrical current also approximates in form an equilateral hyperbola, but differs in its constant time. Apparently, such a regularity of the reaction of living tissue under certain conditions can be broadened to encompass organs and systems (refs. 4, 9), and, as observed in our experiments, even the organism as a whole.

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